

=> fil capl; d que 110

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*inventor
search*

FILE COVERS 1907 - 11 Jun 2002 VOL 136 ISS 24
FILE LAST UPDATED: 9 Jun 2002 (20020609/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

CAS roles have been modified effective December 16, 2001. Please check your SDI profiles to see if they need to be revised. For information on CAS roles, enter HELP ROLES at an arrow prompt or use the CAS Roles thesaurus (/RL field) in this file.

L1	27	SEA	FILE=CAPLUS	ABB=ON	SAKOWICZ R?/AU
L2	986	SEA	FILE=CAPLUS	ABB=ON	GOLDSTEIN L?/AU
L6	7294	SEA	FILE=CAPLUS	ABB=ON	MICROTUBULE/CT
L7	298	SEA	FILE=CAPLUS	ABB=ON	L6(L) (MOTOR# OR PLUS END)
L8	284	SEA	FILE=CAPLUS	ABB=ON	THERMOMYCES LANUGINOS?
L10	6	SEA	FILE=CAPLUS	ABB=ON	L1 AND L2 AND (L7 OR L8)

=> fil medl; d que 143

FILE 'MEDLINE' ENTERED AT 13:10:13 ON 11 JUN 2002

FILE LAST UPDATED: 6 JUN 2002 (20020606/UP). FILE COVERS 1958 TO DATE.

On June 9, 2002, MEDLINE was reloaded. See HELP RLOAD for details.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2002 vocabulary. Enter HELP THESAURUS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

L34	15596	SEA	FILE=MEDLINE	ABB=ON	MICROTUBULE PROTEINS+NT/CT
L35	9	SEA	FILE=MEDLINE	ABB=ON	SAKOWICZ R?/AU
L36	874	SEA	FILE=MEDLINE	ABB=ON	GOLDSTEIN L?/AU
L38	58	SEA	FILE=MEDLINE	ABB=ON	THERMOMYCES LANUGINOSUS
L42	14397	SEA	FILE=MEDLINE	ABB=ON	MICROTUBULES/CT
L43	3	SEA	FILE=MEDLINE	ABB=ON	L35 AND L36 AND (L34 OR L38 OR L42)

=> fil wpids; d que 177

FILE 'WPIDS' ENTERED AT 13:10:14 ON 11 JUN 2002

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FILE LAST UPDATED: 10 JUN 2002 <20020610/UP>
MOST RECENT DERWENT UPDATE 200236 <200236/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> The BATCH option for structure searches has been
enabled in WPINDEX/WPIDS and WPIX >>>

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GUIDES, PLEASE VISIT:
http://www.derwent.com/userguides/dwpi_guide.html <<<

L70 268 SEA FILE=WPIDS ABB=ON MICROTUBULE# OR MICRO TUBULE#
L71 10 SEA FILE=WPIDS ABB=ON SAKOWICZ R?/AU
L72 42 SEA FILE=WPIDS ABB=ON GOLDSTEIN L?/AU
L76 35 SEA FILE=WPIDS ABB=ON THERMOMYCES LANUGINOS?
L77 3 SEA FILE=WPIDS ABB=ON L71 AND L72 AND (L70 OR L76)

=> fil biotechno biotechds; d que 191

FILE 'BIOTECHNO' ENTERED AT 13:10:15 ON 11 JUN 2002
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FILE 'BIOTECHDS' ENTERED AT 13:10:15 ON 11 JUN 2002
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L1 27 SEA FILE=CAPLUS ABB=ON SAKOWICZ R?/AU
L2 986 SEA FILE=CAPLUS ABB=ON GOLDSTEIN L?/AU
L87 7 SEA L1
L88 132 SEA L2
L89 6167 SEA MICROTUBULE# OR MICRO TUBULE#
L90 191 SEA THERMOMYCES LANUGINOS?
L91 3 SEA L87 AND L88 AND (L89 OR L90)

=> fil biosis; d que 1108

FILE 'BIOSIS' ENTERED AT 13:10:17 ON 11 JUN 2002
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 5 June 2002 (20020605/ED)

L101 16 SEA FILE=BIOSIS ABB=ON SAKOWICZ R?/AU
L102 1173 SEA FILE=BIOSIS ABB=ON GOLDSTEIN L?/AU

L103 32776 SEA FILE=BIOSIS ABB=ON MICROTUBULE# OR MICRO TUBULE#
L104 220 SEA FILE=BIOSIS ABB=ON THERMOMYCES LANUGINOS?
L108 7 SEA FILE=BIOSIS ABB=ON L101 AND L102 AND (L103 OR L104)

=> dup rem 143,110,191,1108,177

FILE 'MEDLINE' ENTERED AT 13:10:18 ON 11 JUN 2002

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PROCESSING COMPLETED FOR L43
PROCESSING COMPLETED FOR L10
PROCESSING COMPLETED FOR L91
PROCESSING COMPLETED FOR L108
PROCESSING COMPLETED FOR L77
L116 12 DUP REM L43 L10 L91 L108 L77 (10 DUPLICATES REMOVED)
ANSWERS '1-3' FROM FILE MEDLINE
ANSWERS '4-7' FROM FILE CAPLUS
ANSWER '8' FROM FILE BIOTECHNO
ANSWERS '9-12' FROM FILE BIOSIS

=> d ibib ab 1116 1-12

L116 ANSWER 1 OF 12 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2000095847 MEDLINE
DOCUMENT NUMBER: 20095847 PubMed ID: 10631986
TITLE: Cloning and expression of kinesins from the thermophilic
fungus *Thermomyces lanuginosus*.
AUTHOR: Sakowicz R; Farlow S; Goldstein L S
CORPORATE SOURCE: Howard Hughes Medical Institute, Department of Cellular and
Molecular Medicine, School of Medicine, University of
California, San Diego, La Jolla 92093-0683, USA.
CONTRACT NUMBER: GM35252 (NIGMS)
SOURCE: PROTEIN SCIENCE, (1999 Dec) 8 (12) 2705-10.
Journal code: 9211750. ISSN: 0961-8368.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000229
Last Updated on STN: 20000229
Entered Medline: 20000214

AB The motor domain regions of three novel members of the kinesin superfamily
TLKIF1, TLKIFC, and TLBIMC were identified in a thermophilic fungus
Thermomyces lanuginosus. Based on sequence similarity,
they were classified as members of the known kinesin families Unc104/KIF1,
KAR3, and BIMC. TLKIF1 was subsequently expressed in *Escherichia coli*. The
expression level was high, and the protein was mostly soluble, easy to
purify, and enzymatically active. TLKIF1 is a monomeric kinesin motor,
which in a gliding motility assay displays a robust plus-directed

microtubule movement up to 2 microm/s. The discovery of TLKIF1 also demonstrates that a family of kinesin motors not previously found in fungi may in fact be used in this group of organisms.

L116 ANSWER 2 OF 12 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1998202613 MEDLINE
DOCUMENT NUMBER: 98202613 PubMed ID: 9535660
TITLE: A marine natural product inhibitor of kinesin motors.
AUTHOR: **Sakowicz R**; Berdelis M S; Ray K; Blackburn C L;
Hopmann C; Faulkner D J; **Goldstein L S**
CORPORATE SOURCE: Department of Pharmacology, Division of Cellular and
Molecular Medicine, Howard Hughes Medical Institute,
University of California, San Diego, 9500 Gilman Drive, La
Jolla, CA 92093-0683, USA.
SOURCE: SCIENCE, (1998 Apr 10) 280 (5361) 292-5.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199804
ENTRY DATE: Entered STN: 19980507
Last Updated on STN: 19980507
Entered Medline: 19980428

AB Members of the kinesin superfamily of motor proteins are essential for mitotic and meiotic spindle organization, chromosome segregation, organelle and vesicle transport, and many other processes that require microtubule-based transport. A compound, adociasulfate-2, was isolated from a marine sponge, Haliclona (also known as Adocia) species, that inhibited kinesin activity by targeting its motor domain and mimicking the activity of the microtubule. Thus, the kinesin-microtubule interaction site could be a useful target for small molecule modulators, and adociasulfate-2 should serve as an archetype for specific inhibitors of kinesin functions.

L116 ANSWER 3 OF 12 MEDLINE
ACCESSION NUMBER: 96196874 MEDLINE
DOCUMENT NUMBER: 96196874 PubMed ID: 8612068
TITLE: The muscle in kinesin.
AUTHOR: **Sakowicz R**; **Goldstein L S**
SOURCE: NATURE STRUCTURAL BIOLOGY, (1996 May) 3 (5) 404-7.
Journal code: 9421566. ISSN: 1072-8368.
PUB. COUNTRY: United States
News Announcement
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199606
ENTRY DATE: Entered STN: 19960613
Last Updated on STN: 19960613
Entered Medline: 19960603

L116 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 1
ACCESSION NUMBER: 1999:487304 CAPLUS
DOCUMENT NUMBER: 131:112405
TITLE: Identification and expression of the microtubule motor protein kinesin TL-.gamma.
INVENTOR(S): **Sakowicz, Roman**; **Goldstein, Lawrence S. B.**
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9937659	A1	19990729	WO 1999-US1355	19990122
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
AU 9924648	A1	19990809	AU 1999-24648	19990122
PRIORITY APPLN. INFO.:			US 1998-72361P	A2 19980123
			WO 1999-US1355	W 19990122

AB The invention concerns the isolation of a nucleic acid sequence from **Thermomyces lanuginosus** that encodes the microtubule motor protein kinesin TL-.gamma. with the following properties: the protein's activity includes plus end-directed microtubule motor activity; the protein has a tail domain that has greater than 60% amino acid sequence identity to a TL-.gamma. tail domain as measured using a sequence comparison algorithm; the protein specifically binds to polyclonal antibodies to TL-.gamma.. The invention also concerns antibodies to TL-.gamma., methods for screening biol. active TL-.gamma., and kits for screening. Using PCR and degenerate primers, TL-.gamma. was amplified from **Thermomyces lanuginosus** genomic DNA. The nucleic acid sequence was then used as a probe to isolate a longer TL-.gamma. sequence. Recombinant TL-.gamma. was prepd. in order to test its activity in a microtubule gliding assay. The pET23-TL-.gamma. expression vector was constructed and expressed in E. coli. The kinesin TL-.gamma. protein was isolated, it was very stable retaining 100% activity up to 40.degree. after incubation for 15 min as measured using a microtubule dependent ATPase assay. Freshly prepd. protein was used to assay microtubule gliding activity. Taxol stabilized microtubule seeds brightly labeled with rhodamine were prepd. by incubating a 1:1 ratio of rhodamine labeled bovine brain tubulin; also unlabeled bovine brain tubulin was incorporated into the assay. Flow chambers prepd. were preadsorbed with TL-.gamma. motor protein. A microtubule/ATP mix contg. polarity marked microtubules, taxol, MgATP and an oxygen scavenging system was then flowed into the chamber. Movement of microtubules was monitored at room temp. on a fluorescence microscope fitted with oil immersion objective and a CCD. For TL-.gamma. activity measurement, recombinant TL-.gamma. protein was attached to a glass coverslip using non-specific adhesion, and gliding of polarity marked microtubules contg. brightly fluorescent rhodamine labeled seeds near their minus ends was recorded by time-lapse digital fluorescence microscopy. Microtubules moved with brightly fluorescent seeds leading, indicating that the immobilized TL-.gamma. protein was moving toward microtubule plus ends. No movement was obsd. in the absence of TL-.gamma.. This expt. demonstrates that TL-.gamma. has plus-ended microtubule motor activity.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L116 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
ACCESSION NUMBER: 1999:451195 CAPLUS
DOCUMENT NUMBER: 131:97592
TITLE: Kinesin motor modulators derived from the marine sponge adocia
INVENTOR(S): Goldstein, Lawrence S. B.; Faulkner, David
John; Sakowicz, Roman; Berdelis, Michael S.;

PATENT ASSIGNEE(S): Blackburn, Christine L.; Hopmann, Cordula
SOURCE: The Regents of the University of California, USA
PCT Int. Appl., 73 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9934806	A1	19990715	WO 1999-US321	19990106
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
AU 9921071	A1	19990726	AU 1999-21071	19990106
EP 1049475	A1	20001108	EP 1999-901353	19990106
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI			
US 6207403	B1	20010327	US 1999-226772	19990106
JP 2002500190	T2	20020108	JP 2000-527255	19990106
PRIORITY APPLN. INFO.:			US 1998-70772P P	19980108
			WO 1999-US321 W	19990106

OTHER SOURCE(S): MARPAT 131:97592

AB This invention provides novel compds. derived from a marine sponge, *Adocia* sp., that specifically modulate kinesin activity by targeting the kinesin motor domain and mimicking the activity of a microtubule. The compds. act as potent anti-mitogens and are useful in a wide variety of in vitro and in vivo applications [e.g. in mitigating a variety of pathol. conditions characterized by abnormal cell mitosis].

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L116 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
ACCESSION NUMBER: 1999:194248 CAPLUS
DOCUMENT NUMBER: 130:233824
TITLE: Plus end-directed microtubule motor protein CENP-E required for *Xenopus* chromosome congression
INVENTOR(S): Wood, Kenneth W.; Sakowicz, Roman; Goldstein, Lawrence S. B.; Cleveland, Don W.
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9913061	A1	19990318	WO 1998-US19231	19980910
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,			

FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2303484 AA 19990318 CA 1998-2303484 19980910
AU 9893918 A1 19990329 AU 1998-93918 19980910
AU 745385 B2 20020321
EP 1012249 A1 20000628 EP 1998-947039 19980910

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI

JP 2001526881 T2 20011225 JP 2000-510850 19980910

PRIORITY APPLN. INFO.:

US 1997-58645P P 19970911

WO 1998-US19231 W 19980910

AB The invention provides isolated nucleic acid and amino acid sequences of *Xenopus* centromere-assocd. protein-E (XCENP-E), antibodies to XCENP-E, methods of screening for CENP-E modulators using biol. active CENP-E, and kits for screening for CENP-E modulators. The full-length cDNA sequences of XCENP-E encodes a protein of 2954 amino acids with a predicted mol. mass of 340 kDa. XCENP-E is a member of the kinesin superfamily of motor proteins, and consists of a 500-amino acid globular N-terminal domain contg. a kinesin-like microtubule motor domain linked to a globular tail domain by a region predicted to form a long, discontinuous .alpha.-helical coiled coil. This is the first biol. active CENP-E isolated and, surprisingly and contrary to previous reports, it demonstrates a motor that powers chromosome movement toward microtubule plus ends. Using immunodepletion and antibody addn. to *Xenopus* egg exts., the present invention further demonstrates that CENP-E plays an essential role in congression.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L116 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 6

ACCESSION NUMBER: 1997:724763 CAPLUS

DOCUMENT NUMBER: 128:45048

TITLE: CENP-E is a plus end-directed kinetochore motor required for metaphase chromosome alignment

AUTHOR(S): Wood, Kenneth W.; Sakowicz, Roman; Goldstein, Lawrence S. B.; Cleveland, Don W.

CORPORATE SOURCE: Lab. of Cell Biol., Ludwig Inst. for Cancer Res., Univ. of California at San Diego, La Jolla, CA, 92093-0660, USA

SOURCE: Cell (Cambridge, Massachusetts) (1997), 91(3), 357-366
CODEN: CELLB5; ISSN: 0092-8674

PUBLISHER: Cell Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Mitosis requires dynamic attachment of chromosomes to spindle microtubules. This interaction is mediated largely by kinetochores. During prometaphase, forces exerted at kinetochores, in combination with polar ejection forces, drive congression of chromosomes to the metaphase plate. A major question has been whether kinetochore-assocd. microtubule motors play an important role in congression. Using immunodepletion from and antibody addn. to *Xenopus* egg exts., we show that the kinetochore-assocd. kinesin-like motor protein CENP-E is essential for positioning chromosomes at the metaphase plate. We further demonstrate that CENP-E powers movement toward microtubule plus ends in vitro. These findings support a model in which CENP-E functions in congression to tether kinetochores to dynamic microtubule plus ends.

L116 ANSWER 8 OF 12 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.

ACCESSION NUMBER: 1999:29367197 BIOTECHNO

TITLE: Adociasulfates 1-6, inhibitors of kinesin motor proteins from the sponge *Haliclona* (aka *Adocia*) sp.

AUTHOR: Blackburn C.L.; Hopmann C.; Sakowicz R.; Berdelis M.S.; Goldstein L.S.B.; Faulkner

D.J.
CORPORATE SOURCE: D.J. Faulkner, Scripps Institution of Oceanography,
Univ. of California at San Diego, San Diego, CA
92093-0212, United States.
E-mail: jfaulkner@ucsd.edu
SOURCE: Journal of Organic Chemistry, (23 JUL 1999), 64/15
(5565-5570)
CODEN: JOCEAH ISSN: 0022-3263
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Adociasulfates 1-6 (1-6) were isolated from an extract of the Palauan
sponge Haliclona (aka Adocia) sp. that inhibited the transport of
stabilized **microtubules** by the motor protein kinesin, which was
immobilized on a microscope slide. The structures of adociasulfates 1-6,
the relative stereochemistry of adociasulfates 1, 2, 5, and 6, and the
relative stereochemistry of subunits of adociasulfates 3 and 4 were
determined by interpretation of spectroscopic data. In a quantitative
assay that measures ATP hydrolysis by kinesin, adociasulfates 2 and 6
were the most active.

L116 ANSWER 9 OF 12 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:461909 BIOSIS
DOCUMENT NUMBER: PREV200100461909
TITLE: Kinesin motor modulators derived from the marine sponge
Adocia.
AUTHOR(S): Goldstein, Lawrence S. B.; Faulkner, David John;
Sakowicz, Roman; Berdelis, Michael S.; Blackburn,
Christine L.; Hopmann, Cordula
CORPORATE SOURCE: San Diego, CA USA
ASSIGNEE: The Regents of the University of California
PATENT INFORMATION: US 6207403 March 27, 2001
SOURCE: Official Gazette of the United States Patent and Trademark
Office Patents, (Mar. 27, 2001) Vol. 1244, No. 4, pp. No
Pagination. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB This invention provides novel compounds derived from a marine sponge,
Adocia sp., that specifically modulate kinesin activity by targeting the
kinesin motor domain and mimicking the activity a **microtubule**.
The compounds act as potent anti-mitogens and are useful in a wide variety of
in vitro and in vivo applications.

L116 ANSWER 10 OF 12 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:15470 BIOSIS
DOCUMENT NUMBER: PREV199900015470
TITLE: Study of the orientation of kinesin motors bound to
microtubules using single molecule fluorescence
polarization spectroscopy.
AUTHOR(S): Sosa, H. (1); Peterman, E. J. G.; Dickson, R. M.;
Sakowicz, R.; Moerner, W. E.; Goldstein, L.
G.
CORPORATE SOURCE: (1) Dep. Pharmacology, Univ. Calif., San Diego, CA 92093
USA
SOURCE: Molecular Biology of the Cell, (Nov., 1998) Vol. 9, No.
SUPPL., pp. 28A.
Meeting Info.: 38th Annual Meeting of the American Society
for Cell Biology San Francisco, California, USA December
12-16, 1998 American Society for Cell Biology
. ISSN: 1059-1524.
DOCUMENT TYPE: Conference

LANGUAGE: English

L116 ANSWER 11 OF 12 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1996:278444 BIOSIS

DOCUMENT NUMBER: PREV199699000800

TITLE: The muscle in kinesin.

AUTHOR(S): Sakowicz, Roman; Goldstein, Lawrence S.
B.

CORPORATE SOURCE: Howard Hughes Med. Inst., Div. Cellular Molecular Med.,
Dep. Pharmacology, Univ. Calif. San Diego, 9500 Gilman
Drive, La Jolla, CA 92093-0683 USA

SOURCE: Nature Structural Biology, (1996) Vol. 3, No. 5, pp.
404-407.
ISSN: 1072-8368.

DOCUMENT TYPE: Article

LANGUAGE: English

L116 ANSWER 12 OF 12 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1997:95559 BIOSIS

DOCUMENT NUMBER: PREV199799394762

TITLE: Cloning, expression, and purification of kinesin
superfamily members from the thermophilic fungus.

AUTHOR(S): Sakowicz, R.; Farlow, S.; Goldstein, L. S.
B.

CORPORATE SOURCE: Howard Hughes Med. Inst., Div. Cell. Mol. Med., Dep.
Pharmacol., Univ. Calif. San Diego, 9500 Gilman Dr., La
Jolla, CA 92093-0683 USA

SOURCE: Molecular Biology of the Cell, (1996) Vol. 7, No. SUPPL.,
pp. 215A.
Meeting Info.: Annual Meeting of the 6th International
Congress on Cell Biology and the 36th American Society for
Cell Biology San Francisco, California, USA December 7-11,
1996
ISSN: 1059-1524.

DOCUMENT TYPE: Conference; Abstract; Conference

LANGUAGE: English

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FILE COVERS 1907 - 11 Jun 2002 VOL 136 ISS 24

FILE LAST UPDATED: 9 Jun 2002 (20020609/ED)

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=> d que 113; d que 119; d que 125; d que 132

L6	7294	SEA	FILE=CAPLUS	ABB=ON	MICROTUBULE/CT
L8	284	SEA	FILE=CAPLUS	ABB=ON	THERMOMYCES LANUGINOS?
L13	1	SEA	FILE=CAPLUS	ABB=ON	L6 AND L8

L6	7294	SEA	FILE=CAPLUS	ABB=ON	MICROTUBULE/CT
L9	236153	SEA	FILE=CAPLUS	ABB=ON	SEQUENCE#/CW
L15	542	SEA	FILE=CAPLUS	ABB=ON	TAIL DOMAIN#
L16	204	SEA	FILE=CAPLUS	ABB=ON	TL GAMMA
L19	5	SEA	FILE=CAPLUS	ABB=ON	L6 AND (L15 OR L16) AND L9

L6	7294	SEA	FILE=CAPLUS	ABB=ON	MICROTUBULE/CT
L7	298	SEA	FILE=CAPLUS	ABB=ON	L6(L) (MOTOR# OR PLUS END)
L15	542	SEA	FILE=CAPLUS	ABB=ON	TAIL DOMAIN#
L16	204	SEA	FILE=CAPLUS	ABB=ON	TL GAMMA
L25	5	SEA	FILE=CAPLUS	ABB=ON	L7 AND (L15 OR L16)

L6	7294	SEA	FILE=CAPLUS	ABB=ON	MICROTUBULE/CT
L7	298	SEA	FILE=CAPLUS	ABB=ON	L6(L) (MOTOR# OR PLUS END)
L9	236153	SEA	FILE=CAPLUS	ABB=ON	SEQUENCE#/CW
L20	377186	SEA	FILE=CAPLUS	ABB=ON	EXPRESS?/OBI OR VECTOR#/OBI OR ENCOD?/OBI OR TRANSFECT?/OBI
L22	165625	SEA	FILE=CAPLUS	ABB=ON	ANTIBOD?/OBI
L28	104763	SEA	FILE=CAPLUS	ABB=ON	(DNA OR CDNA OR NUCLEIC ACID# OR RNA OR MRNA) (L) L9
L29	180736	SEA	FILE=CAPLUS	ABB=ON	PROTEIN#(L) L9
L30	75223	SEA	FILE=CAPLUS	ABB=ON	L29 AND L28

L32 8 SEA FILE=CAPLUS ABB=ON L7 AND L30 AND L20 AND L22

=> s (l13 or l19 or l25 or l32) not l10

L117 12 (L13 OR L19 OR L25 OR L32) NOT (L10) *previously printed w/ inventor search*

=> fil medl

FILE 'MEDLINE' ENTERED AT 13:13:04 ON 11 JUN 2002

FILE LAST UPDATED: 6 JUN 2002 (20020606/UP). FILE COVERS 1958 TO DATE.

On June 9, 2002, MEDLINE was reloaded. See HELP RLOAD for details.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2002 vocabulary. Enter HELP THESAURUS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

=> d que 144; d que 146; d que 156; d que 150; d que 154; d que 168

L34 15596 SEA FILE=MEDLINE ABB=ON MICROTUBULE PROTEINS+NT/CT
L38 58 SEA FILE=MEDLINE ABB=ON THERMOMYCES LANUGINOSUS
L42 14397 SEA FILE=MEDLINE ABB=ON MICROTUBULES/CT
L44 1 SEA FILE=MEDLINE ABB=ON (L34 OR L42) AND L38

L34 15596 SEA FILE=MEDLINE ABB=ON MICROTUBULE PROTEINS+NT/CT
L42 14397 SEA FILE=MEDLINE ABB=ON MICROTUBULES/CT
L45 5 SEA FILE=MEDLINE ABB=ON TL GAMMA
L46 0 SEA FILE=MEDLINE ABB=ON (L34 OR L42) AND L45

L34 15596 SEA FILE=MEDLINE ABB=ON MICROTUBULE PROTEINS+NT/CT
L42 14397 SEA FILE=MEDLINE ABB=ON MICROTUBULES/CT
L47 221 SEA FILE=MEDLINE ABB=ON PLUS END
L48 485 SEA FILE=MEDLINE ABB=ON TAIL DOMAIN#
L49 242 SEA FILE=MEDLINE ABB=ON (L34 OR L42) AND (L47 OR L48)
L55 26782 SEA FILE=MEDLINE ABB=ON GENETIC VECTORS/CT
L56 0 SEA FILE=MEDLINE ABB=ON L49 AND L55

L34 15596 SEA FILE=MEDLINE ABB=ON MICROTUBULE PROTEINS+NT/CT
L42 14397 SEA FILE=MEDLINE ABB=ON MICROTUBULES/CT
L47 221 SEA FILE=MEDLINE ABB=ON PLUS END
L48 485 SEA FILE=MEDLINE ABB=ON TAIL DOMAIN#
L50 1 SEA FILE=MEDLINE ABB=ON (L34 OR L42) AND L47 AND L48

L34 15596 SEA FILE=MEDLINE ABB=ON MICROTUBULE PROTEINS+NT/CT
L42 14397 SEA FILE=MEDLINE ABB=ON MICROTUBULES/CT
L48 485 SEA FILE=MEDLINE ABB=ON TAIL DOMAIN#
L51 514123 SEA FILE=MEDLINE ABB=ON D24.611.125./CT = *antibodies*
L54 5 SEA FILE=MEDLINE ABB=ON (L34 OR L42) AND L48 AND L51

L47 221 SEA FILE=MEDLINE ABB=ON PLUS END
L48 485 SEA FILE=MEDLINE ABB=ON TAIL DOMAIN#
L60 260773 SEA FILE=MEDLINE ABB=ON BASE SEQUENCE/CT
L66 595 SEA FILE=MEDLINE ABB=ON MICROTUBULE PROTEINS/CT
L68 2 SEA FILE=MEDLINE ABB=ON L66 AND L60 AND (L47 OR L48)

=> s (144 or 150 or 154 or 168) not 143

L118 7 (L44 OR L50 OR L54 OR L68) NOT (L43) *previously printed w/ inventor search*

=> fil wpids

FILE 'WPIDS' ENTERED AT 13:13:06 ON 11 JUN 2002
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FILE LAST UPDATED: 10 JUN 2002 <20020610/UP>
MOST RECENT DERWENT UPDATE 200236 <200236/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

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enabled in WPINDEX/WPIDS and WPIX >>>

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=> d que 175; d que 181

L75 1 SEA FILE=WPIDS ABB=ON TL GAMMA

L70 268 SEA FILE=WPIDS ABB=ON MICROTUBULE# OR MICRO TUBULE#
L73 8 SEA FILE=WPIDS ABB=ON TAIL DOMAIN#
L74 93 SEA FILE=WPIDS ABB=ON PLUS END
L76 35 SEA FILE=WPIDS ABB=ON THERMOMYCES LANUGINOS?
L81 3 SEA FILE=WPIDS ABB=ON L70 AND (L73 OR L74 OR L76)

=> s (175 or 181) not 177

L119 1 (L75 OR L81) NOT (L77) *previously printed w/ inventor search*

=> fil biotechno biotechds

FILE 'BIOTECHNO' ENTERED AT 13:13:09 ON 11 JUN 2002
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=> d que 195; d que 192; d que 196; d que 1100

L89 6167 SEA MICROTUBULE# OR MICRO TUBULE#
L90 191 SEA THERMOMYCES LANUGINOS?
L95 1 SEA L89 AND L90

L92 1 SEA TL GAMMA

L89 6167 SEA MICROTUBULE# OR MICRO TUBULE#
L93 315 SEA TAIL DOMAIN#
L94 77 SEA PLUS END
L96 1 SEA L89 AND L93 AND L94

L89 6167 SEA MICROTUBULE# OR MICRO TUBULE#
L93 315 SEA TAIL DOMAIN#
L94 77 SEA PLUS END
L99 167397 SEA ENCOD?
L100 11 SEA L89 AND (L93 OR L94) AND L99

=> s (195 or 192 or 196 or 1100) not 191

L120 12 (L95 OR L92 OR L96 OR L100) NOT (L91)

=> fil biosis

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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 5 June 2002 (20020605/ED)

=> d que 1109;d que 1111; d que 1112;d que 1115

L103 32776 SEA FILE=BIOSIS ABB=ON MICROTUBULE# OR MICRO TUBULE#
L105 4 SEA FILE=BIOSIS ABB=ON TL GAMMA
L109 1 SEA FILE=BIOSIS ABB=ON L105 AND L103

L103 32776 SEA FILE=BIOSIS ABB=ON MICROTUBULE# OR MICRO TUBULE#
L104 220 SEA FILE=BIOSIS ABB=ON THERMOMYCES LANUGINOS?
L111 2 SEA FILE=BIOSIS ABB=ON L103 AND L104

L103 32776 SEA FILE=BIOSIS ABB=ON MICROTUBULE# OR MICRO TUBULE#
L106 560 SEA FILE=BIOSIS ABB=ON TAIL DOMAIN#
L107 259 SEA FILE=BIOSIS ABB=ON PLUS END
L112 1 SEA FILE=BIOSIS ABB=ON L103 AND L106 AND L107

L103 32776 SEA FILE=BIOSIS ABB=ON MICROTUBULE# OR MICRO TUBULE#
L106 560 SEA FILE=BIOSIS ABB=ON TAIL DOMAIN#

L107 259 SEA FILE=BIOSIS ABB=ON PLUS END
L113 169566 SEA FILE=BIOSIS ABB=ON ENCOD?
L114 730 SEA FILE=BIOSIS ABB=ON L103(3A)MOTOR
L115 9 SEA FILE=BIOSIS ABB=ON L114 AND L113 AND (L106 OR L107)

=> s (l109 or l111 or l112 or l115) not l108

L121 9 (L109 OR L111 OR L112 OR L115) NOT L108 *previously printed w/ inventer search*

=> dup rem l118,l117,l120,l121,l119

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FILE 'CAPLUS' ENTERED AT 13:13:50 ON 11 JUN 2002

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FILE 'WPIDS' ENTERED AT 13:13:50 ON 11 JUN 2002

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PROCESSING COMPLETED FOR L118

PROCESSING COMPLETED FOR L117

PROCESSING COMPLETED FOR L120

PROCESSING COMPLETED FOR L121

PROCESSING COMPLETED FOR L119

L122 30 DUP REM L118 L117 L120 L121 L119 (11 DUPLICATES REMOVED)
ANSWERS '1-7' FROM FILE MEDLINE
ANSWERS '8-18' FROM FILE CAPLUS
ANSWERS '19-28' FROM FILE BIOTECHNO
ANSWER '29' FROM FILE BIOSIS
ANSWER '30' FROM FILE WPIDS

=> d ibib ab 1-30; fil hom

L122 ANSWER 1 OF 30 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 95014709 MEDLINE
DOCUMENT NUMBER: 95014709 PubMed ID: 7929562
TITLE: A novel microtubule-based motor protein (KIF4) for organelle transports, whose expression is regulated developmentally.
AUTHOR: Sekine Y; Okada Y; Noda Y; Kondo S; Aizawa H; Takemura R; Hirokawa N
CORPORATE SOURCE: Department of Anatomy and Cell Biology, School of Medicine, University of Tokyo, Japan.
SOURCE: JOURNAL OF CELL BIOLOGY, (1994 Oct) 127 (1) 187-201.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D12646
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941031

AB To understand the mechanisms of transport for organelles in the axon, we isolated and sequenced the cDNA encoding KIF4 from murine brain, and

characterized the molecule biochemically and immunocytochemically. Complete amino acid sequence analysis of KIF4 and ultrastructural studies of KIF4 molecules expressed in Sf9 cells revealed that the protein contains 1,231 amino acid residues (M(r) 139,550) and that the molecule (116-nm rod with globular heads and tail) consists of three domains: an NH2-terminal globular motor domain, a central alpha-helical stalk domain and a COOH-terminal **tail domain**. KIF4 protein has the property of nucleotide-dependent binding to microtubules, microtubule-activated ATPase activity, and microtubule **plus-end**-directed motility. Northern blot analysis and in situ hybridization demonstrated that KIF4 is strongly expressed in juvenile tissues including differentiated young neurons, while its expression is decreased considerably in adult mice except in spleen. Immunocytochemical studies revealed that KIF4 colocalized with membranous organelles both in growth cones of differentiated neurons and in the cytoplasm of cultured fibroblasts. During mitotic phase of cell cycle, KIF4 appears to colocalize with membranous organelles in the mitotic spindle. Hence we conclude that KIF4 is a novel microtubule-associated anterograde motor protein for membranous organelles, the expression of which is regulated developmentally.

L122 ANSWER 2 OF 30 MEDLINE
ACCESSION NUMBER: 2000227826 MEDLINE
DOCUMENT NUMBER: 20227826 PubMed ID: 10762698
TITLE: Cdk5 and MAPK are associated with complexes of cytoskeletal proteins in rat brain.
COMMENT: Erratum in: Brain Res Mol Brain Res 2000 Aug 14;80(1):109
Erratum in: Veeranna GJ [corrected to Veeranna]
AUTHOR: Veeranna; Shetty K T; Takahashi M; Grant P; Pant H C
CORPORATE SOURCE: Laboratory of Neurochemistry, National Institutes of Neurological Disorders and Stroke, National Institutes of Health, Bldg. 36, Rm. 4D20, Bethesda, MD 20892-4130, USA.
SOURCE: BRAIN RESEARCH. MOLECULAR BRAIN RESEARCH, (2000 Mar 29) 76 (2) 229-36.
Journal code: 8908640. ISSN: 0169-328X.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000616
Last Updated on STN: 20010604
Entered Medline: 20000605

AB Neurofilament proteins, the major cytoskeletal components of large myelinated axons, are highly phosphorylated by second messenger-dependent and -independent kinases. These kinases, together with tubulins and other cytoskeletal proteins, have been shown to bind to neurofilament preparations. Cdk5 and Erk2, proline-directed kinases in neuronal tissues, phosphorylate the Lys-Ser-Pro (KSP) repeats in **tail domains** of NF-H, NF-M, and other axonal proteins such as tau and synapsin. In neurofilament and microtubule preparations from rat brain, we demonstrated by Western blot analysis that cdk5, a neuronal cyclin dependent kinase and Erk1/2 were associated with complexes of NF proteins, tubulins and tau. Using P13(suc1) affinity chromatography, a procedure known to bind cdc2-like kinases in proliferating cells with high affinity, we obtained a P13 complex from a rat brain extract exhibiting the same profiles of cdk5 and Erk2 bound to cytoskeletal proteins. The phosphorylation activities of these preparations and the effect of the cdk5 inhibitor, butyrolactone, were consistent with the presence of active kinases. Finally, during a column fractionation and purification of Erk kinases from rat brain extracts, fractions enriched in Erk kinase activity also exhibited co-elution of phosphorylated NF-H, tubulin, tau and cdk5. We suggest that in mammalian brain, different kinases, their regulators

and phosphatases form multimeric complexes with cytoskeletal proteins and regulate multisite phosphorylation from synthesis in the cell body to transport and assembly in the axon.

L122 ANSWER 3 OF 30 MEDLINE
ACCESSION NUMBER: 1999034594 MEDLINE
DOCUMENT NUMBER: 99034594 PubMed ID: 9817761
TITLE: Light chain-dependent regulation of Kinesin's interaction with microtubules.
AUTHOR: Verhey K J; Lizotte D L; Abramson T; Barenboim L; Schnapp B J; Rapoport T A
CORPORATE SOURCE: Howard Hughes Medical Institute, Harvard Medical School, Boston, Massachusetts 02115, USA.
SOURCE: JOURNAL OF CELL BIOLOGY, (1998 Nov 16) 143 (4) 1053-66.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981221

AB We have investigated the mechanism by which conventional kinesin is prevented from binding to microtubules (MTs) when not transporting cargo. Kinesin heavy chain (HC) was expressed in COS cells either alone or with kinesin light chain (LC). Immunofluorescence microscopy and MT cosedimentation experiments demonstrate that the binding of HC to MTs is inhibited by coexpression of LC. Association between the chains involves the LC NH2-terminal domain, including the heptad repeats, and requires a region of HC that includes the conserved region of the stalk domain and the NH2 terminus of the **tail domain**. Inhibition of MT binding requires in addition the COOH-terminal 64 amino acids of HC. Interaction between the tail and the motor domains of HC is supported by sedimentation experiments that indicate that kinesin is in a folded conformation. A pH shift from 7.2 to 6.8 releases inhibition of kinesin without changing its sedimentation behavior. Endogenous kinesin in COS cells also shows pH-sensitive inhibition of MT binding. Taken together, our results provide evidence that a function of LC is to keep kinesin in an inactive ground state by inducing an interaction between the tail and motor domains of HC; activation for cargo transport may be triggered by a small conformational change that releases the inhibition of the motor domain for MT binding.

L122 ANSWER 4 OF 30 MEDLINE
ACCESSION NUMBER: 97428309 MEDLINE
DOCUMENT NUMBER: 97428309 PubMed ID: 9281579
TITLE: Kinesin- and myosin-driven steps of vesicle recruitment for Ca2+-regulated exocytosis.
AUTHOR: Bi G Q; Morris R L; Liao G; Alderton J M; Scholey J M; Steinhardt R A
CORPORATE SOURCE: Department of Molecular and Cell Biology, University of California, Berkeley, California 94720-3200, USA.
SOURCE: JOURNAL OF CELL BIOLOGY, (1997 Sep 8) 138 (5) 999-1008.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971021
Last Updated on STN: 19980206
Entered Medline: 19971009

AB Kinesin and myosin have been proposed to transport intracellular organelles and vesicles to the cell periphery in several cell systems. However, there has been little direct observation of the role of these motor proteins in the delivery of vesicles during regulated exocytosis in intact cells. Using a confocal microscope, we triggered local bursts of Ca^{2+} -regulated exocytosis by wounding the cell membrane and visualized the resulting individual exocytotic events in real time. Different temporal phases of the exocytosis burst were distinguished by their sensitivities to reagents targeting different motor proteins. The function blocking antikinesin antibody SUK4 as well as the stalk-tail fragment of kinesin heavy chain specifically inhibited a slow phase, while butanedione monoxime, a myosin ATPase inhibitor, inhibited both the slow and fast phases. The blockage of Ca^{2+} /calmodulin-dependent protein kinase II with autoinhibitory peptide also inhibited the slow and fast phases, consistent with disruption of a myosin-actin- dependent step of vesicle recruitment. Membrane resealing after wounding was also inhibited by these reagents. Our direct observations provide evidence that in intact living cells, kinesin and myosin motors may mediate two sequential transport steps that recruit vesicles to the release sites of Ca^{2+} -regulated exocytosis, although the identity of the responsible myosin isoform is not yet known. They also indicate the existence of three semistable vesicular pools along this regulated membrane trafficking pathway. In addition, our results provide in vivo evidence for the cargo-binding function of the kinesin heavy chain **tail domain**.

L122 ANSWER 5 OF 30 MEDLINE

ACCESSION NUMBER: 95151739 MEDLINE

DOCUMENT NUMBER: 95151739 PubMed ID: 7849016

TITLE: Comparison of the motile and enzymatic properties of two microtubule minus-end-directed motors, ncd and cytoplasmic dynein.

AUTHOR: Shimizu T; Toyoshima Y Y; Edamatsu M; Vale R D

CORPORATE SOURCE: National Institute of Bioscience and Human-Technology, Ibaraki, Japan.

CONTRACT NUMBER: 38499

SOURCE: BIOCHEMISTRY, (1995 Feb 7) 34 (5) 1575-82.

Journal code: 0370623. ISSN: 0006-2960.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199503

ENTRY DATE: Entered STN: 19950322

Last Updated on STN: 19950322

Entered Medline: 19950313

AB Cytoplasmic dynein and ncd, a kinesin-related protein from *Drosophila*, are motor proteins that move toward the minus ends of microtubules, while kinesin moves to the microtubule **plus end**. In previous work, we examined the nucleotide dependence of motility and enzymatic activity by kinesin [Shimizu, T., Furusawa, K., Ohashi, S., Toyoshima, Y. Y., Okuno, M., Malik, F., & Vale, R. D., (1991) *J. Cell Biol.* 112, 1189-1197]. In this study, we examined these activities of the cytoplasmic dynein from bovine brain and ncd in order to explore what enzymatic features might be shared by these two minus-end-directed motors. Both ncd and cytoplasmic dynein demonstrated an activation of ATPase activity upon the addition of microtubules (30-fold and 6-fold, respectively). A significant difference between ncd and cytoplasmic dynein was their relative sensitivity to vanadate and to aluminum fluoride. In contrast to cytoplasmic dynein, ncd polypeptide was not cleaved by UV-vanadate treatment, and its ATPase and motility were unaffected by vanadate (up to 0.1 mM). When the nucleotide requirement for movement as examined using a battery of 20 nucleotides and nucleotide analogues, cytoplasmic dynein was found to exhibit a specificity very similar to that of axonemal dyneins

from Tetrahymena. Surprisingly, however, the nucleotide specificities of in vitro motility produced by ncd or its construct, GST/MC1 (a fusion protein of glutathione S-transferase and 210-700 of the predicted ncd amino acid sequence), were quite distinct from that of kinesin. Thus, the nucleotide specificity profiles of members of the kinesin motor superfamily do not appear to be identical.

L122 ANSWER 6 OF 30 MEDLINE

ACCESSION NUMBER: 94043030 MEDLINE

DOCUMENT NUMBER: 94043030 PubMed ID: 8226779

TITLE: Interaction of the **tail domain** of high molecular weight subunits of neurofilaments with the COOH-terminal region of tubulin and its regulation by tau protein kinase II.

AUTHOR: Miyasaka H; Okabe S; Ishiguro K; Uchida T; Hirokawa N

CORPORATE SOURCE: Department of Anatomy and Cell Biology, Faculty of Medicine, University of Tokyo, Japan.

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1993 Oct 25) 268 (30) 22695-702.

Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199312

ENTRY DATE: Entered STN: 19940117

Last Updated on STN: 20020420

Entered Medline: 19931201

AB We previously showed that neurofilaments interact with microtubules (MTs) via their high molecular weight subunits (NF-H) after alkaline phosphatase treatment. Here we studied the effects of phosphorylation of NF-H on this interaction. tau protein kinase II, Ser/Thr protein kinase, phosphorylated NF-H in the **tail domain**, decreased its electrophoretic mobility to a native level, and also restored its property to be less interactive with MTs. Phosphorylation by cAMP-dependent protein kinase caused no shift of electrophoretic mobility or dissociation from MTs. We conclude that the **tail domain** of NF-H directly interacts with the MT surface, and the interaction is regulated via phosphorylation of the **tail domain** of NF-H by Ser/Thr protein kinase like tau protein kinase II. To characterize the binding domain of NF-H on MTs, subtilisin digestion of MTs and competition analysis with the MT binding fragment of tau protein were performed. The dissociation constant of NF-H to subtilisin MTs was higher than that to intact MTs. The maximum binding of NF-H was reduced when tau fragments existed. These results revealed that the COOH-terminal region of tubulin is involved in the binding to NF-H, and the NF-H and microtubule-associated protein binding domains are closely apposed on the surface of MTs.

L122 ANSWER 7 OF 30 MEDLINE

ACCESSION NUMBER: 90153980 MEDLINE

DOCUMENT NUMBER: 90153980 PubMed ID: 2137456

TITLE: The primary structure and analysis of the squid kinesin heavy chain.

AUTHOR: Kosik K S; Orecchio L D; Schnapp B; Inouye H; Neve R L

CORPORATE SOURCE: Department of Neurology (Neuroscience), Harvard Medical School, Boston, Massachusetts.

CONTRACT NUMBER: AG06172 (NIA)

AG06601 (NIA)

NS20824 (NINDS)

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1990 Feb 25) 265 (6) 3278-83.

Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-J05258
ENTRY MONTH: 199003
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19900601
Entered Medline: 19900323

AB We report the cDNA sequence of the squid kinesin heavy chain and compared the predicted amino acid sequence with that of the *Drosophila* heavy chain as reported by Yang, J.T., Laymon, R.A., and Goldstein, L.S. B. (1989) Cell 56, 879-889). We compared the two kinesin sequences with regard to the predicted physicochemical parameters of hydrophobicity, charge, and propensities of the secondary conformations. A comparison of the sequences from the two species reveals the head, stalk, and tail domains because a reduced degree of conservation demarcates the stalk. The charge profile indicates that the head region is nearly neutral, the stalk region acidic, and the tail is basic. The Fourier transform analysis of the hydrophobic profile of the stalk shows predominant peaks at 1/3.5 and 1/2.3, which are indexed as the second and third orders of the period 7 residue. As in the *Drosophila* sequence, the rod domain is divided into an amino and a carboxyl subdomain by a predicted hinge region. We show that the disposition of hydrophobic residues is distinct in these two subdomains. In particular, the heptad repeat is more regular in the amino-terminal rod domain than in the carboxyl-terminal rod domain. The tail region is positively charged, a feature that is consistent with the known electrostatic interaction between the heavy chain and negatively charged surfaces such as glass coverslips and latex beads. Three monoclonal antibodies to the kinesin heavy chain have been mapped to a region within the carboxyl terminus of the stalk.

L122 ANSWER 8 OF 30 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 6
ACCESSION NUMBER: 1993:186069 CAPLUS
DOCUMENT NUMBER: 118:186069
TITLE: Cloning and expression of a human kinesin heavy chain gene: interaction of the carboxy-terminal domain with cytoplasmic microtubules in transfected CV-1 cells
AUTHOR(S): Navone, Francesca; Niclas, Joshua; Hom-Booher, Nora; Sparks, Lynne; Bernstein, Harris D.; McCaffrey, Gretchen; Vale, Ronald D.
CORPORATE SOURCE: Dep. Pharmacol., Univ. California, San Francisco, CA, 94143, USA
SOURCE: J. Cell Biol. (1992), 117(6), 1263-75
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To understand the interactions between the microtubule-based motor protein kinesin and intracellular components, the kinesin heavy chain and its different domains were expressed in CV-1 monkey kidney epithelial cells and their distributions were examd. by immunofluorescence microscopy. The cDNAs encoding the kinesin heavy chain were cloned and sequenced from a human placental library. The human kinesin heavy chain exhibits a high level of sequence identity to the previously cloned invertebrate kinesin heavy chains; homologies between the COOH-terminal domain of human and invertebrate kinesins and the nonmotor domain of the *Aspergillus* kinesin-like protein bimC were also found. The gene encoding the human kinesin heavy chain also contains a small upstream open reading frame in a G-C rich 5' untranslated region, features that are assocd. with translational regulation in certain mRNAs. After transient expression in CV-1 cells, the kinesin heavy chain showed both a diffuse distribution and a filamentous staining pattern that coaligned with microtubules but not

vimentin intermediate filaments. Altering the no. and distribution of microtubules with taxol or nocodazole produced corresponding changes in the localization of the expressed kinesin heavy chain. The expressed NH2-terminal motor and the COOH-terminal **tail domains**, but not the .alpha.-helical coiled-coil rod domain, also colocalized with microtubules. The finding that both the kinesin motor and **tail domains** can interact with cytoplasmic microtubules raises the possibility that kinesin could crossbridge and induce sliding between microtubules under certain circumstances.

L122 ANSWER 9 OF 30 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:123032 CAPLUS
DOCUMENT NUMBER: 136:178998
TITLE: Novel human kinesin motor protein HsKip3d and cDNA and therapeutic use
INVENTOR(S): Beraud, Christophe; Freedman, Richard
PATENT ASSIGNEE(S): Cytokinetics, Inc., USA
SOURCE: PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002012268	A1	20020214	WO 2001-US24285	20010803
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
US 6391601	B1	20020521	US 2000-724511	20001127
PRIORITY APPLN. INFO.:			US 2000-632344	A 20000803
AB	The invention provides isolated nucleic acid and amino acid sequences of HsKip3d, antibodies to HsKip3d, methods of screening for HsKip3d modulators using biol. active HsKip3d, and kits for screening for HsKip3d modulators. The mRNA expression profile in various tissues are also provided.			
REFERENCE COUNT:	2	THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT		

L122 ANSWER 10 OF 30 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:345890 CAPLUS
DOCUMENT NUMBER: 136:336317
TITLE: Novel human kinesin motor protein HsKif21b and cDNA and therapeutic use
INVENTOR(S): Beraud, Christophe; Freedman, Richard
PATENT ASSIGNEE(S): Cytokinetics, Inc., USA
SOURCE: U.S., 33 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6383796	B1	20020507	US 2000-718692	20001122

AB The invention provides isolated nucleic acid and amino acid sequences of HsKif21b, antibodies to HsKif21b, methods of screening for HsKif21b modulators using biol. active HsKif21b, and kits for screening for HsKif21b modulators. The mRNA expression profile in various tissues are also provided.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L122 ANSWER 11 OF 30 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:327864 CAPLUS

DOCUMENT NUMBER: 136:320421

TITLE: Novel human kinesin motor protein HsKrp5 and cDNA and therapeutic use

INVENTOR(S): Beraud, Christophe; Freedman, Richard

PATENT ASSIGNEE(S): Cytokinetics, Inc., USA

SOURCE: U.S., 29 pp., Cont. of U.S. Ser. No. 641,807.

CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6379941	B1	20020430	US 2000-724517	20001127
PRIORITY APPLN. INFO.:			US 2000-641807	A1 20000817

AB The invention provides isolated nucleic acid and amino acid sequences of HsKrp5, antibodies to HsKrp5, methods of screening for HsKrp5 modulators using biol. active HsKrp5, and kits for screening for HsKrp5 modulators. The mRNA expression profile in various tissues are also provided.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L122 ANSWER 12 OF 30 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:6347 CAPLUS

DOCUMENT NUMBER: 136:81691

TITLE: Sequence, characterization and use of a novel human kinesin motor protein HsKifc2

INVENTOR(S): Beraud, Christophe; Freedman, Richard

PATENT ASSIGNEE(S): Cytokinetics, Inc., USA

SOURCE: U.S., 27 pp.
CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6335189	B1	20020101	US 2000-721383	20001122

AB The invention provides isolated cDNA and amino acid sequences of a novel human kinesin motor protein, HsKifc2, antibodies to HsKifc2, methods of screening for HsKifc2 modulators using biol. active HsKifc2, and kits for screening for HsKifc2 modulators. The kinesin HsKifc2 comprises a motor domain and has microtubule-stimulated ATPase activity. The qual. tissue expression profile of HsKifc2 in variety of tissues is shown.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L122 ANSWER 13 OF 30 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:935617 CAPLUS

DOCUMENT NUMBER: 136:49424

TITLE: Novel human kinesin motor protein HsKif17 and cDNA and

therapeutic use
INVENTOR(S): Beraud, Christophe; Freedman, Richard
PATENT ASSIGNEE(S): Cytokinetics, Inc., USA
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001098314	A2	20011227	WO 2001-US19811	20010620

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-597602 A 20000620

AB The invention provides isolated nucleic acid and amino acid sequences of HsKif17, antibodies to HsKif17, methods of screening for HsKif17 modulators using biol. active HsKif17, and kits for screening for HsKif17 modulators. The mRNA expression profile in various tissues are also provided.

L122 ANSWER 14 OF 30 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:924028 CAPLUS

DOCUMENT NUMBER: 136:50048

TITLE: cDNA **encoding** a kinesin motor protein
HsKip3a and its use in the treatment of cell proliferation disorders

INVENTOR(S): Beraud, Christophe; Craven, Andrew; Yu, Ming;
Sakowicz, Roman; Patel, Umesh A.; Davies, Katherine A.

PATENT ASSIGNEE(S): Cytokinetics, Inc., USA

SOURCE: PCT Int. Appl., 70 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001096593	A2	20011220	WO 2001-US19308	20010615

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-594655 A 20000615

AB The invention provides isolated nucleic acid and amino acid sequences of a novel human kinesin motor protein HsKip3a. Also provided are antibodies to HsKip3a and methods of screening for HsKip3a modulators using biol. active HsKip3a (having ATPase activity), and kits for screening for HsKip3a modulators. Methods of screening for HsKip3a ATPase activity in a

multi-well plate as part of a high-throughput screen are provided. Modulators of HsKip3a can be used in the treatment of cancer, hyperplasias, restenosis, cardiac hypertrophy, immune disorders and inflammation.

L122 ANSWER 15 OF 30 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:933089 CAPLUS
DOCUMENT NUMBER: 136:50049
TITLE: Novel human kinesin motor protein HsKif16a and cDNA and therapeutic use
INVENTOR(S): Beraud, Christophe; Freedman, Richard
PATENT ASSIGNEE(S): Cytokinetics, Inc., USA
SOURCE: U.S., 27 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
	US 6333184	B1	20011225	US 2000-718841	20001122
AB	The invention provides isolated nucleic acid and amino acid sequences of HsKif16a, antibodies to HsKif16a, methods of screening for HsKif16a modulators using biol. active HsKif16a, and kits for screening for HsKif16a modulators. The mRNA expression profile in various tissues and cell lines are also provided.				

L122 ANSWER 16 OF 30 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:765527 CAPLUS
DOCUMENT NUMBER: 132:74953
TITLE: Kinesin's processivity results from mechanical and chemical coordination between the ATP hydrolysis cycles of the two motor domains
AUTHOR(S): Hancock, William O.; Howard, Jonathon
CORPORATE SOURCE: Department of Physiology and Biophysics, University of Washington, Seattle, WA, 98195-7290, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1999), 96(23), 13147-13152
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Kinesin is a processive motor protein: A single mol. can walk continuously along a microtubule for several micrometers, taking hundreds of 8-nm steps without dissocg. To elucidate the biochem. and structural basis for processivity, we have engineered a heterodimeric one-headed kinesin and compared its biochem. properties to those of the wild-type two-headed mol. Our construct retains the functionally important neck and **tail domains** and supports motility in high-d. microtubule gliding assays, though it fails to move at the single-mol. level. We find that the ATPase rate of one-headed kinesin is 3-6 s⁻¹ and that detachment from the microtubule occurs at a similar rate (3 s⁻¹). This establishes that one-headed kinesin usually detaches once per ATP hydrolysis cycle. Furthermore, we identify the rate-limiting step in the one-headed hydrolysis cycle as detachment from the microtubule in the ADP.cntdot.Pi state. Because the ATPase and detachment rates are roughly an order of magnitude lower than the corresponding rates for two-headed kinesin, the detachment of one head in the homodimer (in the ADP.cntdot.Pi state) must be accelerated by the other head. We hypothesize that this results from internal strain generated when the second head binds. This idea accords with a hand-over-hand model for processivity in which the release of the trailing head is contingent on the binding of the forward head. These new

results, together with previously published ones, allow us to propose a pathway that defines the chem. and mech. cycle for two-headed kinesin.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L122 ANSWER 17 OF 30 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:714869 CAPLUS

DOCUMENT NUMBER: 132:105554

TITLE: Identification of kinesin-C, a calmodulin-binding carboxy-terminal kinesin in animal (*Strongylocentrotus purpuratus*) cells

AUTHOR(S): Rogers, Gregory C.; Hart, Cynthia L.; Wedaman, Karen P.; Scholey, Jonathan M.

CORPORATE SOURCE: Section of Molecular and Cellular Biology, University of California, Davis, CA, 95616, USA

SOURCE: Journal of Molecular Biology (1999), 294(1), 1-8
CODEN: JMOBAK; ISSN: 0022-2836

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Several novel members of the kinesin superfamily, until now identified only in plants, are unique in their ability to bind calmodulin in the presence of Ca^{2+} . Here, we identify the 1st such kinesin in an animal system. Sequence anal. of this new motor, called kinesin-C, predicts that it is a large C-terminal kinesin, 1624 amino acid residues in length, with a predicted mol. mass of 181 kDa. Kinesin-C is predicted to contain a kinesin motor domain at its C-terminus, linked to a segment of .alpha.-helical coiled-coil 950 amino acid residues long, ending with an N-terminal proline-rich **tail domain**. A putative calmodulin-binding domain resides at the extreme C-terminus of the motor polypeptide, and recombinant kinesin-C binds to a calmodulin-affinity column in a Ca^{2+} -dependent fashion. The presence of this novel calmodulin-binding motor in sea urchin embryos suggests that it plays a crit. role in Ca^{2+} -dependent events during early sea urchin development.
(c) 1999 Academic Press.

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L122 ANSWER 18 OF 30 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1993:554334 CAPLUS

DOCUMENT NUMBER: 119:154334

TITLE: Sequence and structure of a new coiled coil protein from a microtubule bundle in *Giardia*

AUTHOR(S): Marshall, Jonathan; Holberton, David V.

CORPORATE SOURCE: Dep. Life Sci., Nottingham Univ., Nottingham, NG7 2RD, UK

SOURCE: J. Mol. Biol. (1993), 231(2), 521-30
CODEN: JMOBAK; ISSN: 0022-2836

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The flagellate *Giardia* is reportedly (and controversially) the most primitive eukaryotic organism known. The trophozoite is without organelles but has an extensive cytoskeleton of microtubules and assocd. structural proteins. Overlapping cDNAs and genomic DNAs were cloned and sequenced for a 101,000 Mr protein involved in microtubule bundling in the median body of *G. lamblia* cells. The polypeptide chain appears to be mainly .alpha.-helical with the repeating amphipathic heptapeptides characteristic of a coiled coil mol., but without homol. to known microtubule-assocd. proteins. Domain anal. suggests a structure in which a rod of three linked coils spans 695 residues (.apprx.103 nm), with the ends of the chain forming compact globular head and **tail domains** of .apprx.11 kDa and .apprx.7 kDa. The rod domain has internal sequence repeats of 24 residues caused by multiple phase shifts

in the coiled coil heptapeptide positions. These repeats have a conserved side-chain pattern which contributes the most significant periodicities in Fourier transforms of the linear distributions of apolar and charged residues. The best alignment of the pattern has 21 complete repeats of 24 residues and 9 partial repeats of 21 or fewer residues. The apolar residue phase shifts will produce a regular stutter in the hydrophobic core of the coiled coil. This structure is reminiscent of .beta.-giardin, another coiled coil protein with a broken seam found in the Giardia cytoskeleton. Although the underlying sequence motif is different for the two proteins, the general feature of being regularly divided into segments might relate to a similar mechanism of interaction with microtubules.

L122 ANSWER 19 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.DUPLICATE

ACCESSION NUMBER: 1999:29462572 BIOTECHNO

TITLE: A novel mouse kinesin of the UNC-104/KIF1 subfamily
encoded by the Kif1b gene

AUTHOR: Gong T.-W.L.; Winnicki R.S.; Kohrman D.C.; Lomax M.I.

CORPORATE SOURCE: M.I. Lomax, Kresge Hearing Research Institute, Dept.
Otolaryngology/Head Neck Surg., University of
Michigan, Ann Arbor, MI 48109, United States.
E-mail: mlomax@umich.edu

SOURCE: Gene, (1999), 239/1 (117-127), 33 reference(s)

CODEN: GENED6 ISSN: 0378-1119

PUBLISHER ITEM IDENT.: S0378111999003704

DOCUMENT TYPE: Journal; Article

COUNTRY: Netherlands

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Kinesin and kinesin-related proteins are **microtubule**-dependent motor proteins that transport organelles. We have cloned and sequenced a full-length 9924 bp mouse cDNA for a new kinesin of the UNC-104/KIF1 subfamily. Northern blot analysis of mouse RNAs detected high levels of a 10 kb mRNA in brain and eye, but lower levels in other tissues. Human RNA dot-blot analysis detected this mRNA in all tissues examined, although at different levels. The overall structure of the new kinesin (predicted size 204 kDa) was most similar to mouse KIF1A; however, 2.1 kb of the 5' portion of the cDNA were identical to the published sequence for KIF1B (Nangaku, M., Sato-Yoshitake, R., Okada, Y., Noda, Y., Takemura, R., Yamazaki, H., Hirokawa, N., 1994. KIF1B, a novel **microtubule plus end**-directed monomeric motor protein for transport of mitochondria. Cell 79, 1209-1220). We localized the Kif1b gene to the distal end of mouse Chromosome 4 by haplotype analysis of an interspecific backcross from The Jackson Laboratory. We had previously mapped the gene for the novel kinesin to the same location (Gong, T.-W.L., Burmeister, M., Lomax, M.I., 1996b. The novel gene D4Mille maps to mouse Chromosome 4 and human Chromosome 1p36. Mamm. Genome 7, 790-791). We conclude, therefore, that the Kif1b gene generates two major kinesin isoforms by alternative splicing. The shorter 7.8 kb mRNA **encodes** a 130 kDa kinesin, KIF1Bp130, whereas the 10 kb mRNA **encodes** a 204 kDa kinesin, KIF1Bp204. In addition, alternative splicing of two exons in the conserved region adjacent to the motor domain generates four different isoforms of each kinesin, leading to eight kinesin isoforms derived from the Kif1b gene. (C) 1999 Elsevier Science B.V. All rights reserved.

L122 ANSWER 20 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.DUPLICATE

ACCESSION NUMBER: 1998:28373627 BIOTECHNO

TITLE: A developmentally regulated kinesin-related motor
protein from Dictyostelium discoideum

AUTHOR: De Hostos E.L.; McCaffrey G.; Sucgang R.; Pierce D.W.;
Vale R.D.

CORPORATE SOURCE: E.L. De Hostos, Dept. of Biochemistry and Cell Biol.,
Rice University, Houston, TX 77005, United States.

SOURCE: E-mail: hostos@bioc.rice.edu
Molecular Biology of the Cell, (1998), 9/8
(2093-2106), 80 reference(s)
CODEN: MBCEEV ISSN: 1059-1524

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The cellular slime mold Dictyostelium discoideum is an attractive system for studying the roles of **microtubule**-based motility in cell development and differentiation. In this work, we report the first molecular characterization of kinesin-related proteins (KRPs) in Dictyostelium. A PCR-based strategy was used to isolate DNA fragments **encoding** six KRPs, several of which are induced during the developmental program that is initiated by starvation. The complete sequence of one such developmentally regulated KRP (designated K7) was determined and found to be a novel member of the kinesin superfamily. The motor domain of K7 is most similar to that of conventional kinesin, but unlike conventional kinesin, K7 is not predicted to have an extensive .alpha.- helical coiled-coil domain. The nonmotor domain is unusual and is rich in Asn, Gln, and Thr residues; similar sequences are found in other developmentally regulated genes in Dictyostelium. K7, expressed in Escherichia coli, supports **plus end**-directed **microtubule** motility in vitro at a speed of 0.14 .mu.m/s, indicating that it is a bona fide motor protein. The K7 motor is found only in developing cells and reaches a peak level of expression between 12 and 16 h after starvation. By immunofluorescence microscopy, K7 localizes to a membranous perinuclear structure. To examine K7 function, we prepared a null cell line but found that these cells show no gross developmental abnormalities. However, when cultivated in the presence of wild-type cells, the K7-null cells are mostly absent from the prestalk zone of the slug. This result suggests that in a population composed largely of wild-type cells, the absence of the K7 motor protein interferes either with the ability of the cells to localize to the prestalk zone or to differentiate into prestalk cells.

L122 ANSWER 21 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.DUPLICATE
ACCESSION NUMBER: 1995:25151564 BIOTECHNO
TITLE: Characterization of a minus end-directed kinesin-like motor protein from cultured mammalian cells
AUTHOR: Kuriyama R.; Kofron M.; Essner R.; Kato T.;
Dragas-Granoic S.; Omoto C.K.; Khodjakov A.
CORPORATE SOURCE: Dept. of Cell Biology/Neuroanatomy, 4-135 Jackson
Hall, University of Minnesota, 321 Church Street
SE, Minneapolis, MN 05545, United States.
SOURCE: Journal of Cell Biology, (1995), 129/4 (1049-1059)
CODEN: JCLBA3 ISSN: 0021-9525

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Using the CHO2 monoclonal antibody raised against CHO spindles (Sellitto, C., M. Kimble, and R. Kuriyama. 1992. Cell Motil. Cytoskeleton. 22:7-24) we identified a 66-kD protein located at the interphase centrosome and mitotic spindle. Isolated cDNAs for the antigen **encode** a 622-amino acid polypeptide. Sequence analysis revealed the presence of 340-amino acid residues in the COOH terminus, which is homologous to the motor domain conserved among other members of the kinesin superfamily. The protein is composed of a central .alpha.- helical portion with globular domains at both NH.sub.2 and COOH termini, and the epitope to the monoclonal antibody resides in the central .alpha.-helical stalk. A series of deletion constructs were created for in vitro analysis of **microtubule** interactions. While the **microtubule** binding

and bundling activities require both the presence of the COOH terminus and the .alpha.-helical domain, the NH.sub.2-terminal half of the antigen lacked the ability to interact with **microtubules**. The full-length as well as deleted proteins consisting of the COOH-terminal motor and the central .alpha.-helical stalk supported **microtubule** gliding, with velocity ranging from 1.0 to 8.4 .mu.m/minute. The speed of **microtubule** movement decreased with decreasing lengths of the central stalk attached to the COOH-terminal motor. The **microtubules** moved with their **plus end** leading, indicating that the antigen is a minus end- directed motor. The CHO2 sequence shows 86% identify to HSET, a gene located at the centromeric end of the human MHC region in chromosome 6 (Ando, A., Y. Y. Kikuti, H. Kawata, N. Okamoto, T. Imai, T. Eki, K. Yokoyama, E. Soeda, T. Ikemura, K. Abe, and H. Inoko. 1994. Immunogenetics. 39:194-200), indicating that HSET might represent a human homologue of the CHO2 antigen.

L122 ANSWER 22 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.DUPLICATE
ACCESSION NUMBER: 1994:24355991 BIOTECHNO
TITLE: Characterization of the KLP68D kinesin-like protein in Drosophila: Possible roles in axonal transport
AUTHOR: Pesavento P.A.; Stewart R.J.; Goldstein L.S.B.
CORPORATE SOURCE: Department of Pharmacology, Howard Hughes Medical Institute, University of California, 9500 Gilman Drive, San Diego, CA 92093-0683, United States.
SOURCE: Journal of Cell Biology, (1994), 127/4 (1041-1048)
CODEN: JCLBA3 ISSN: 0021-9525
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB This paper describes the molecular and biochemical properties of KLP68D, a new kinesin-like motor protein in Drosophila melanogaster. Sequence analysis of a full-length cDNA **encoding** KLP68D demonstrates that this protein has a domain that shares significant sequence identity with the entire 340-amin acid kinesin heavy chain motor domain. Sequences extending beyond the motor domain predict a region of alpha-helical coiled-coil followed by a globular 'tail' region; there is significant sequence similarity between the alpha- helical coiled-coil region of the KLP68D protein and similar regions of the KIF3 protein of mouse and the KRP85 protein of sea urchin. This finding suggests that all three proteins may be members of the same family, and that they all perform related functions. KLP68D protein produced in Escherichia coli is, like kinesin itself, a **plus-end** directed **microtubule** motor. In situ hybridization analysis of KLP68D RNA in Drosophila embryos indicates that the KLP68D gene is expressed primarily in the central nervous system and in a subset of the peripheral nervous system during embryogenesis. Thus, KLP68D may be used for anterograde axonal transport and could conceivably move cargoes in fly neurons different than those moved by kinesin heavy chain or other **plus-end** directed motors.

L122 ANSWER 23 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.DUPLICATE
ACCESSION NUMBER: 1992:22315277 BIOTECHNO
TITLE: A **plus-end**-directed motor enzyme that moves antiparallel **microtubules** in vitro localizes to the interzone of mitotic spindles
AUTHOR: Nislow C.; Lombillo V.A.; Kuriyama R.; McIntosh J.R.
CORPORATE SOURCE: Dept. of Mol., Cell./Developm. Biol., University of Colorado, Boulder, CO 80309, United States.
SOURCE: Nature, (1992), 359/6395 (543-547)
CODEN: NATUAS ISSN: 0028-0836
DOCUMENT TYPE: Journal; Article

COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Mitosis comprises a complex set of overlapping motile events, many of which involve **microtubule**-dependent motor enzymes. Here we describe a new member of the kinesin superfamily. The protein was originally identified as a spindle antigen by the CHO1 monoclonal antibody and shown to be required for mitotic progression. We have cloned the gene that **encodes** this antigen and found that its sequence contains a domain with strong sequence similarity to the motor domain of kinesin-like proteins. The product of this gene, expressed in bacteria, can cross-bridge antiparallel **microtubules** in vitro, and in the presence of Mg-ATP, **microtubules** slide over one another in a fashion reminiscent of **microtubule** movements during spindle elongation.

L122 ANSWER 24 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.DUPLICATE

ACCESSION NUMBER: 1990:21038274 BIOTECHNO
TITLE: The kinesin-like ncd protein of Drosophila is a minus end-directed **microtubule** motor

AUTHOR: McDonald H.B.; Stewart R.J.; Goldstein L.S.B.
CORPORATE SOURCE: Department of Cellular and Developmental Biology, Harvard University, Cambridge, MA 02138, United States.

SOURCE: Cell, (1990), 63/6 (1159-1165)
CODEN: CELLB5 ISSN: 0092-8674

DOCUMENT TYPE: Journal; Article

COUNTRY: United States

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The Drosophila ncd gene is required for chromosome segregation during female meiosis. Previous analyses suggested that the ncd gene **encoded** a protein with sequence similarity to the kinesin motor domain, which suggested that, like kinesin, the ncd protein might be a **plus end-directed microtubule** motor. Here we describe the expression of ncd protein in E. coli and the initial characterization of the ncd protein's motor properties. The ncd protein is indeed a **microtubule** motor, but the polarity of movement is minus end directed. The ncd protein also has **microtubule** bundling activity. These findings limit possible models for the in vivo functions of the ncd protein and suggest that motor proteins with similar sequence can generate movement in opposite directions along a **microtubule**.

L122 ANSWER 25 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.

ACCESSION NUMBER: 2001:32288523 BIOTECHNO
TITLE: Rab27a enables myosin Va-dependent melanosome capture by recruiting the myosin to the organelle

AUTHOR: Wu X.; Rao K.; Bowers M.B.; Copeland N.G.; Jenkins N.A.; Hammer III J.A.

CORPORATE SOURCE: J.A. Hammer III, Laboratory of Cell Biology, National Heart Lung/Blood Institute, National Institutes of Health, Bethesda, MD 20892, United States.

E-mail: hammerj@nhlbi.nih.gov
SOURCE: Journal of Cell Science, (2001), 114/6 (1091-1100), 39 reference(s)

CODEN: JNCSAI ISSN: 0021-9533

DOCUMENT TYPE: Journal; Article

COUNTRY: United Kingdom

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The peripheral accumulation of melanosomes characteristic of wild-type mouse melanocytes is driven by a cooperative process involving

long-range, bidirectional, **microtubule**-dependent movement coupled to capture and local movement in the actin-rich periphery by myosin Va, the product of the dilute locus. Genetic evidence suggests that Rab27a, the product of the ashen locus, functions with myosin Va in this process. Here we show that ashen melanocytes, like dilute melanocytes, exhibit normal dendritic morphology and melanosome biogenesis, an abnormal accumulation of end-stage melanosomes in the cell center, and rapid, bidirectional, **microtubule**-dependent melanosome movements between the cell center and the periphery. This phenotype suggests that ashen melanocytes, like dilute melanocytes, are defective in peripheral melanosome capture. Consistent with this, introduction into ashen melanocytes of cDNAs **encoding** wild-type and GTP-bound versions of Rab27a restores the peripheral accumulation of melanosomes in a **microtubule**-dependent manner. Conversely, introduction into wild-type melanocytes of the GDP-bound version of Rab27a generates an ashen/dilute phenotype. Rab27a colocalizes with endstage melanosomes in wild-type cells, and is most concentrated in melanosome-rich dendritic tips, where it also colocalizes with myosin Va. Finally, neither endogenous myosin Va nor an expressed, GFP-tagged, myosin Va **tail domain** fusion protein colocalize with melanosomes in ashen melanocytes, in contrast to that seen previously in wild-type cells. These results argue that Rab27a serves to enable the myosin Va-dependent capture of melanosomes delivered to the periphery by bidirectional, **microtubule**-dependent transport, and that it does so by recruiting the myosin to the melanosome surface. We suggest that Rab27a, in its GTP-bound and melanosome-associated form, predominates in the periphery, and that it is this form that recruits the myosin, enabling capture. These results argue that Rab27a serves as a myosin Va 'receptor', and add to the growing evidence that Rab GTPases regulate vesicle motors as well as SNARE pairing.

L122 ANSWER 26 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.
ACCESSION NUMBER: 2000:32182219 BIOTECHNO
TITLE: Properties of the nonhelical end domains of vimentin suggest a role in maintaining intermediate filament network structure
AUTHOR: Lowrie D.J. Jr.; Stickney J.T.; Ip W.
CORPORATE SOURCE: W. Ip, Department of Cell Biology, University of Cincinnati, College of Medicine, P.O. Box 670521, Cincinnati, OH 45267-0521, United States.
E-mail: wallace.ip@uc.edu
SOURCE: Journal of Structural Biology, (2000), 132/2 (83-94), 83 reference(s)
CODEN: JSBIEM ISSN: 1047-8477
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB To investigate the functional role of the nonhelical domains of the intermediate filament (IF) protein vimentin, we carried out transient transfection of constructs **encoding** fusion proteins of these domains with enhanced green fluorescent protein (EGFP). Expression of these fusion proteins did not have any effect on the endogenous IF networks of transfected cells. However, the head domain-EGFP fusion protein localized almost exclusively to the nucleus. This localization could be disrupted in a reversible fashion by chilling cells. Furthermore, the head domain was capable of targeting to the nucleus a strictly cytoplasmic protein, pyruvate kinase. Thus, the vimentin head domain contains information that specifically directs proteins into the nucleus. In contrast, the nonhelical **tail domain** of vimentin, when expressed as a fusion protein with EGFP, was retained in the cytoplasm. Cytoplasmic retention of **tail domain**-containing fusion proteins appeared to be dependent on the integrity of

the **microtubule** network. Our results are consistent with a proposal that the nonhelical end domains of vimentin are involved in maintaining an extended IF network by exerting oppositely directed forces along the filaments. The head domains exert a nuclear-directed force while the **tail domains** extend the IF network toward the cell periphery via a **microtubule**-dependent mechanism.
.COPYRG. 2000 Academic Press.

L122 ANSWER 27 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.
ACCESSION NUMBER: 1998:28083275 BIOTECHNO
TITLE: Cloning and expression of chicken CLIP-170 and Restin isoforms
AUTHOR: Griparic L.; Volosky J.M.; Keller T.C.S. III
CORPORATE SOURCE: T.C.S. Keller III, Molecular Biophysics Program,
Florida State University, Tallahassee, FL 32306-4370,
United States.
E-mail: tkeller@bio.fsu.edu
SOURCE: Gene, (1998), 206/2 (195-208), 44 reference(s)
CODEN: GENED6 ISSN: 0378-1119
PUBLISHER ITEM IDENT.: S0378111997005854
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English

AB We have cloned cDNA for the chicken homologues of human CLIP-170 and Restin and characterized expression of chicken CLIP-170 and Restin messages in a variety of chicken tissues. Chicken CLIP-170 and Restin, like the human homologues, differ only in a stretch of 35 amino acids present in Restin but missing from CLIP-170. This Restin-specific insert is perfectly conserved between the chicken and human sequences at both the protein and nucleotide level and contributes an additional five heptads to one of the heptad repeat regions in the central α -helical coiled-coil rod domain. Other highly conserved chicken and human CLIP-170/Restin regions confirm the importance of certain protein domains as crucial for protein function, including two CAP-Gly **microtubule**-binding motifs in the N-terminal globular head domain and two CCHC metal-binding motifs in the C-terminal globular **tail domain**. We have used Southern DNA blot analysis and PCR amplification of exon-intron junctions of chicken genomic DNA to confirm that CLIP-170 and Restin are isoforms **encoded** by the same gene. Semiquantitative RT-PCR analysis of CLIP-170 and Restin mRNA expression revealed expression of both isoforms in a variety of chicken tissues but in different ratios. In the tissues tested, except brain, the message for CLIP-170 was more abundant than that for Restin. Comparison of the levels of CLIP-170 and Restin messages in RNA from chicken and human intestinal epithelial cells revealed remarkably similar ratios in the two species. Our data suggest that expression of CLIP-170 and Restin is differentially regulated and that the two isoforms have distinct functions in a wide variety of cells.

L122 ANSWER 28 OF 30 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.
ACCESSION NUMBER: 1998:28219671 BIOTECHNO
TITLE: cDNA cloning and expression analysis of genes encoding GSH synthesis in roots of the heavy-metal accumulator Brassica juncea L.: Evidence for Cd-induction of a putative mitochondrial γ -glutamylcysteine synthetase isoform
AUTHOR: Schafer H.J.; Haag-Kerwer A.; Rausch T.
CORPORATE SOURCE: T. Rausch, Botanisches Institut, Ruprecht-Karls-Universität, Im Neuenheimer Feld 360, 69120 Heidelberg, Germany.
SOURCE: Plant Molecular Biology, (1998), 37/1 (87-97), 53 reference(s)

CODEN: PMBIDB ISSN: 0167-4412
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English

AB In roots of *Brassica juncea* L. cadmium (Cd) exposure (25/.mu.M) induces a massive formation of phytochelatins (PCs), which is accompanied by an only moderate decrease (-20%) of the putative PC precursor glutathione (GSH). As PC formation in roots could be the result of local GSH de novo synthesis and/or depend on GSH import from the shoot, we have analyzed the expression of the enzymes involved in GSH synthesis in the root, namely OAS(thiol)lyase (OAS-TL; catalysing the last step in Cys biosynthesis), .gamma.-glutamylcysteine synthetase (.gamma.-ECS), and glutathione synthetase (GSHS). cDNA clones were isolated from a cDNA library prepared from heavy metal exposed roots. Protein sequences from cDNA clones encoding OAS-TL, .gamma.-ECS, and GSHS, all exhibited putative mitochondrial targeting sequences, however, for OAS-TL also two putative cytosolic isoforms were isolated. Furthermore, we have cloned several metallothionein cDNAs of the MT2 group. Northern blot analysis with coding region probes revealed that in roots of Cd-exposed plants transcript amounts for OAS-TL and GSHS were only moderately increased, whereas .gamma.-ECS mRNA showed a stronger increase. Expression analysis with 3'-UTR probes indicated that among the putative mitochondrial OAS-TL, .gamma.-ECS and GSHS isoforms only .gamma.-ECS was up-regulated in response to Cd exposure. Conversely, transcripts for MT2 appeared to be slightly reduced. The results indicate that in roots Cd-induced PC synthesis correlates with a moderate increase of expression of genes involved in GSH synthesis, the change for .gamma.-ECS being most pronounced.

L122 ANSWER 29 OF 30 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1999:56314 BIOSIS

DOCUMENT NUMBER: PREV199900056314

TITLE: Heterotrimeric kinesin II is the **microtubule motor** protein responsible for pigment dispersion in *Xenopus* melanophores.

AUTHOR(S): Tuma, M. Carolina; Zill, Andrew; Le Bot, Nathalie; Vernos, Isabelle; Gelfand, Vladimir (1)

CORPORATE SOURCE: (1) Univ. Illinois, Dep. Cell Structural Biol., B107 Chemical Life Sciences Lab., 601 S. Goodwin Ave., Urbana, IL 61801 USA

SOURCE: Journal of Cell Biology, (Dec. 14, 1998) Vol. 143, No. 6, pp. 1547-1558.
ISSN: 0021-9525.

DOCUMENT TYPE: Article

LANGUAGE: English

AB Melanophores move pigment organelles (melanosomes) from the cell center to the periphery and vice-versa. These bidirectional movements require cytoplasmic microtubules and microfilaments and depend on the function of microtubule motors and a myosin. Earlier we found that melanosomes purified from *Xenopus* melanophores contain the **plus end microtubule motor** kinesin II, indicating that it may be involved in dispersion (Rogers, S.L., I.S. Tint, P.C. Fanapour, and V.I. Gelfand. 1997. Proc. Natl. Acad. Sci. USA. 94: 3720-3725). Here, we generated a dominant-negative construct **encoding** green fluorescent protein fused to the stalk-tail region of *Xenopus* kinesin-like protein 3 (Xklp3), the 95-kD motor subunit of *Xenopus* kinesin II, and introduced it into melanophores. Overexpression of the fusion protein inhibited pigment dispersion but had no effect on aggregation. To control for the specificity of this effect, we studied the kinesin-dependent movement of lysosomes. Neither dispersion of lysosomes in acidic conditions nor their clustering under alkaline conditions was affected by the mutant Xklp3. Furthermore, microinjection of melanophores with SUK4, a

function-blocking kinesin antibody, inhibited dispersion of lysosomes but had no effect on melanosome transport. We conclude that melanosome dispersion is powered by kinesin II and not by conventional kinesin. This paper demonstrates that kinesin II moves membrane-bound organelles.

L122 ANSWER 30 OF 30 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2000-116370 [10] WPIDS
DOC. NO. CPI: C2000-035514
TITLE: Novel proteins and nucleic acids e.g. for treating neurodegeneration.
DERWENT CLASS: B04 D16 K08
INVENTOR(S): BOGAERT, T A O E; DE RAEYMAEKER, M C; GEYSEN, J J G H; LUYTEN, W H M L; MAERTENS, L J S; VAN DE CRAEN, M; VERHASSELT, P; MAERTEN, L J S
PATENT ASSIGNEE(S): (JANC) JANSSEN PHARM NV
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9963080	A1	19991209	(200010)*	EN	146
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9943735	A	19991220	(200021)		
EP 1092019	A1	20010418	(200123)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9963080	A1	WO 1999-EP3848	19990602
AU 9943735	A	AU 1999-43735	19990602
EP 1092019	A1	EP 1999-926511	19990602
		WO 1999-EP3848	19990602

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 9943735	A Based on	WO 9963080
EP 1092019	A1 Based on	WO 9963080

PRIORITY APPLN. INFO: GB 1998-11962 19980603

AB WO 9963080 A UPAB: 20000228

NOVELTY - Vertebrate protein homolog (I) of a UNC-53 protein of *Caenorhabditis elegans*, contains at least one of 8 amino acid (aa) sequence blocks, or conservative mutants, is encoded by a 9.6 or 7.7 kb sequence (or by their functional equivalents) or has an aa sequence of about 2380, 1830 or 2430 nucleotides, or their variants or truncated forms. All sequences fully defined in the specification.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) cDNA (II) encoding (I);
- (2) nucleic acid (IIa) that hybridizes to (II) under highly stringent conditions;
- (3) DNA expression vector containing (II);
- (4) host cells transformed or transfected with the vector of (3);

(5) transgenic cells, tissues and organs containing a transgene to express (I);

(6) production of a mutant non-human vertebrate, with altered cell behavior, regulation of cell motility or shape, or direction of migration, by introducing a mutation into the wild-type gene for (I);

(7) pharmaceutical composition containing (I) or (II) plus carrier, diluent or excipient;

(8) method for identifying compounds (A) that inhibit or enhance regulation of cell behavior, growth, shape or motility, or the direction of cell migration, comprising contacting the compound with a host cell of (4) and screening for a phenotype;

(9) (A) identified this way and compositions containing them;

(10) method for identifying compounds (B) that inhibit or enhance transcription of a (I)-encoding gene, comprising contacting the compound with a host cell of (4) and monitoring the level of the reporter molecule and comparing the results to a control;

(11) (B) identified this way and composition containing them;

(12) kits for methods (8) and (10);

(13) methods for identifying vertebrate homologs of the unc-53 gene, comprising hybridizing to a cDNA library a suitable oligonucleotide sequence of 15-50 nucleotides of the nucleic acid sequence encoding UNC-53, under conditions which allow the identification of homologous sequences;

(14) methods for identifying a protein (C) active in a cell transduction pathway that involves (I), comprising contacting an extract of the cell with the vertebrate homolog of an UNC-53 protein of C. elegans, identifying any vertebrate homolog of UNC-53 protein/protein complex formed, and analyzing the complex to identify the bound protein;

(15) (C) identified this way and composition containing them;

(16) production of (I) by culturing cells of (4);

(17) method for identifying compounds (D) that inhibit or enhance expression of (I), comprising contacting a cell expressing the homolog with the compound and monitoring for a phenotype change;

(18) composition containing (D);

(19) assay for determining expression of (I) or of the gene encoding it, comprising contacting a cell or extract of it, with an antibody to the homolog, linked to a reporter molecule and monitoring for the presence of the reporter molecule;

(20) method for identifying compounds (E) that inhibit or enhance association of (I) with **microtubules** or their plus ends, comprising contacting the compound with a transgenic cell, tissue or organism expressing UNC-53 protein or vertebrate homolog, which is operably linked to a reporter molecule and screening for the localization of the reporter molecule as compared to a control;

(21) (E) and compositions containing them;

(22) kits for method (20);

(23) targeting a protein to a cell **microtubule**, or its **plus end** region;

(24) method for identifying compounds (F) that covalently modify (I), comprising contacting a cell extract from a cell expressing the homolog with a mixture of enzymes comprising candidate modifying enzymes in the presence of an inhibitor or covalent modification of a protein, identifying any covalently modified UNC-53 protein and identifying the molecule involved in the modification step; and

(25) method for identifying compounds (G) that reduce or enhance toxicity of (I), comprising contacting the compound with a cell, tissue or organism of (5), and monitoring for the presence of the reporter molecule adjacent to the **microtubules** or the plus regions of them; and

(26) the plasmids pG13303 and pG13305, deposited as LMBP 3936 and 3937, respectively.

ACTIVITY - Anticancer; anti-neurodegeneration; antifibrotic; anti-adhesive; antisclerotic; antimetastatic; anti-arthritis.

MECHANISM OF ACTION - (I) binds to microtubules or their plus ends.

USE - (I), also nucleic acid (II) that encodes it, are used to promote neural regeneration, revascularization and wound healing; also for treating neurodegenerative disease, acute traumatic injury, fibrotic disease and autoimmune diseases (e.g. rheumatoid arthritis and sclerosis). (II) can also be used for recombinant production of (I), as a source of probes for detecting allelic variants and polymorphisms, for sequencing genomic DNA and for detecting (I) expression; and as source of therapeutic antisense sequences. Cells that express (I) are used to identify regulators of cell shape, growth, motility and migration (potentially useful in the same way as (I), or for inhibiting spread of disease-inducing cells, metastasis, loss of contact inhibition and cancer). They can also be used to identify proteins that are involved in signal transduction pathways also involving (I) (useful in the same way as (I)), and to identify compounds that alter attachment of (I) to microtubules. A target gene coupled to a (I)-encoding sequence may be used to deliver the target gene to a cellular microtubule or its plus ends.

ADVANTAGE - None given.

Dwg.0/17

FILE 'HOME' ENTERED AT 13:14:09 ON 11 JUN 2002